Blood Pressure, Glycemia, and Body Habitus among a sample of African Americans in Central Ohio

Undergraduate Research Thesis

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Abstract

Chronic degenerative conditions increase as physiological function declines over the life span. Today, coronary artery and cerebrovascular diseases, malignant neoplasms, and noninsulin-dependent diabetes mellitus are leading causes of morbidity and mortality across affluent and underdeveloped societies. Elevated blood pressure, plasma glucose, and body mass index (BMI) are risk factors for these outcomes. In the USA, African Americans show more cardiovascular disease, diabetes, hypertension, and obesity than European Americans. One reason for such disparities may be physiological differences, while others may include sociocultural differences. To explore these influences, we examine blood pressure, glycemia, fat patterning, and body habitus among a sample of 114 older middle-class African Americans residing in Central Ohio during 1995. Our results indicate that BMI and waist/hip ratio positively associated with elevated post-load glucose. Additionally, elevated post-load glucose was significantly associated with upper arm circumference and triceps, subscapular and suprailiac skinfolds. Subscapular skinfolds and abdominal depth were significantly associated with elevated fasting glucose. Abdominal depth predicted both elevated fasting and post-load glucose as well as systolic hypertension. SBP predicted elevated fasting and post-load glucose in our sample. However, no measured variable was significantly related to diastolic hypertension. Our participants represent middle-class African Americans residing in Central Ohio. Their fasting glucose, two-hour post-load glucose, BMI, and wait-hip ratio are above cut-points suggesting diabetes and obesity. These results support the conclusion that such risk factors affect those of higher socioeconomic status as well as the more commonly portrayed poorer classes of African Americans.

Introduction

Chronic health problems and diseases increase as physiological function declines over the life span. Today, coronary artery and cerebrovascular diseases, malignant neoplasms (cancers), and noninsulin-dependent diabetes mellitus are leading causes of morbidity and mortality across most societies, both affluent and less affluent (Crews and Ice 2012; Heron 2013; Murphy et al. 2013; Zimmet et al. 2001). A spectrum of pre-existing risk factors for chronic conditions such as cardiovascular diseases including elevated blood pressure, plasma glucose, and body mass have been identified (Angell et al. 2008; Bartley et al. 2014; WHO 1985; Zhang et al. 2009; Zimmet et al. 2001). A cluster of such risk factors underlie the life style and metabolism-related condition known as Syndrome X (see Morley 2004). As yet, it is not clear how individual and population variation in this suite of risks develops.

Extensive evidence of long-term disparities in health across "racial/ethnic" divisions within the United States also exists (CDC 2013; NCHS 2009-2012). In particular, African Americans suffer a heavier burden of chronic degenerative conditions than do European Americans (CDC 2013). Obesity, hypertension, cardiovascular diseases, and diabetes all are more prevalent among African Americans than European Americans (CDC 2013; NCHS 2009-2012). Today, 70% of African American men are overweight (BMI $\geq$ 25.0) with 38% obese (BMI $\geq$ 30.0), while 82% of women are overweight with 57% obese; additionally, 18.3% of African Americans have diabetes (NCHS 2009-2012). While obesity often co-occurs with type II diabetes mellitus, African Americans show more diabetes within all weight categories than do European Americans (Zhang et al. 2009). Large proportions of African American men (42%) and women (44%) are hypertensive (BP $\geq$ 140/90) and/or taking antihypertensive medications (NCHS2009-2012). Hypertension-related mortality risk among African Americans is twice that

of European Americans (Angell et al. 2008). Despite heavier burdens of hypertension and hypertension-related mortality risk, Yazdanshenas et al. (2014) found that about one third of hypertensive low-income elderly African Americans from Los Angeles were not treated consistently according to accepted clinical guidelines.

Compared with European Americans, African Americans show poorer health across multiple indicators of high risk for future morbidity and mortality from chronic diseases (Dressler 1993; Heron 2013; Kingston and Smith 1997; Murphy et al. 2013; Rogers et al. 2000). Cardiovascular disease, for example, accounts for 30% of the excessive mortality experienced by African compared to European Americans (Lloyd-Jones et al. 2009). Among African Americans age 55 years and older, neoplasms, cardiovascular and cerebrovascular diseases, and type II diabetes mellitus are the leading causes of death (Murphy et al. 2013). African Americans are also more likely to live in larger households, have lower employment rates, smoke cigarettes and have less healthy diets than European Americans (Rogers et al. 2000). Much of this combination of sociocultural risk factors is related to differences in social class across both African and European Americans (Rogers et al. 2000).

Much previous research among African Americans has been conducted among inner-city and rural samples and those of lower SES (Williams et al. 2010). Such results suggest large differences in health between African and European Americans that may be mainly related to class differences in income and household size. To our knowledge, little research has been reported addressing these risk factors among middle-class African American communities. Therefore, we obtained a sample of 114 African Americans ages 26 to 82 years residing in predominantly middle-class African American neighborhoods and cities of Central Ohio (Columbus, Dayton, Xenia, and Wilberforce Township) during 1995. Our goal was to assess

cardiovascular risk factors (blood pressure, glycemia, body habitus, age, and sex) in a sample less affected by poverty, low SES, or racial discrimination and compare them to previous samples (Curtis et al. 1998; Davis et al. 2014; NCHS 2009-2012; Quarells et al. 2012)

Background

Researchers across medicine, biology, physiology, and bioanthropology have attempted to explain the epidemic of chronic diseases (e.g. cardiovascular diseases, cancer, diabetes) in modern societies. Rising morbidity and mortality from such conditions is a recent development in human life-history evolution. An evolutionary perspective aids in explaining biological causes of chronic conditions and their proximate mechanisms. Williams and Nesse (1991) suggest "diseases of civilization" are attributable to either senescent biology revealed following reduced early-life mortality and increased life spans in recent generations, or mismatches between humankind's current environment and that experienced by our evolutionary forbearers.

As recently as 1900, a newborn in the USA could expect to live only 49 years (Rogers, et al. 2000). In 2010, they could expect to survive almost 80 years (Arias 2014; Rogers et al. 2000). Recent life span increases across populations are secondary to improved survival at all ages, but largely due to better survival at early ages. During later decades of the 20th and into the 21st centuries, survival at ages past 65 years has shown significant increases (Arias 2014). As later-life survival has increased, rates of morbidity and mortality from chronic conditions have risen among the oldest members of populations. It is doubtful our evolutionary ancestors lived sufficiently long to experience the physiological declines and range of degenerative symptoms associated with chronic diseases of the 21st century (Caspari and Lee 2006; Crews and Ice 2012).

Obviously, increased life expectancies contributed to a universal rise in chronic diseases among people surviving to their latter decades of life.

Another suggestion is that rising chronic health conditions result from mismatches between our current and evolutionary environments. Some suggest we are genetically adapted to consume a "Paleolithic diet" similar to what our evolutionary ancestors utilized (Eaton et al. 1988). These contributions suggest a diet of raw, unprocessed foods rich in lean proteins and low in processed carbohydrates is the perfect human diet. They then attribute our current burden of cardiovascular diseases to deviation from this "Paleolithic diet." It is likely some genetic components contribute to an evolutionary mismatch between our current and previous diets. However, such a reductionst view of human kind being adapted to but a single environment is a narrow view of our mosaic evolution across multiple locally variant dietary and resource availability settings and the recent evolution of alleles such at the lactose locus (Laland et al. 2010).

A pleiotropic genotype model offers a nuanced understanding of evolutionary trade-offs and mismatches without dismissing human variation. Williams (1957) argues that pleiotropic genes with opposing effects on the soma may both promote and limit late-life senescence. Pleiotropic traits that decrease fitness during growth/development or peak reproduction years are selected against. Conversely, any traits conferring disadvantages or advantages after reproductive age do not experience strong selection. Thus, traits conferring an early-life advantage, regardless of their effects on late-life survival, are retained in the genome. Later, Neel (1962, 1999) proposed the human genome contains "thrifty" genes predisposing individuals to efficiently utilize scarce resources in their environment. Different human populations evolved in a variety of environments with variable resources and seasonal availabilities thereof. That these differences

have produced broad variation in allele frequencies across human populations is well established with respect to disease-promoting alleles at the ACE, HLA, and FAD1 loci (Mielke et al. 2010).

According to Neel, during times of resource scarcity, individuals who were able to store energy in times of plenty were able to better survive and reproduce during periods of resource scarcity. Combining these models, Crews and Gerber (1999) suggest thifty/pleiotropic alleles may influence chronic conditions by promoting accumulation of specific nutrients previously scarce among human diets, but abundant in modern environments. Although Williams and Nesse (1991) posed longevity and mismatches with the environment of evolutionary adaption as different explanations for chronic conditions, both may work in concert to promote disease in modern societies.

Ethnic Health Disparities

Many conjecture as to why health disparities exist between different groups in the USA. Most often cited are sociocultural differences between African and European American in socioeconomic status (SES), incomes, behaviors, and experiences of discrimination. (CDC 2013). Recent research on genetic differences between socially-defined racial groups in the USA has not supported the conjecture of large biological differences between African and European Americans (reviewed by Crews and Gerber 2008). However, discrimination is a salient feature of African American life in a predominantly European American society (Carlson and Chambrlain 2005; Dressler 1993; Dressler and Bindon 2000). Similarly, average SES is lower among African than European Americans across much of the US, and may affect disease risk and environmental exposures (Rogers et al. 2000).

Numerous studies have examined relationships between ethnicity, biological indicators of health, and SES (D'Anna et al. 2010; Kingston and Smith 1997; Krieger and Sidney 1996; Manton et al. 1987; Menchik 1993; Piper et al. 2015; Sellers et al. 2009; Wang and Beydoun 2007). One hypothesis is that overrepresentation of African Americans in lower SES groups may partly explain observed health disparities between USA ethnic groups. However, even when controlling for SES in large, representative national surveys like the Health and Retirement Survey (Kingston and Smith 1997) and the National Longitudinal Mortality Study (Sorlie and Rogot 1992), African Americans still exhibit higher mortality rates, hypertension, and type II diabetes mellitus than do European Americans. Allen et al. (2014) reported subjective SES was associated with cardiovascular disease risk in European, but not among African American participants. When examining mortality disparities between European and African Americans, Rogers et al. (2000) reported observed disparities disappeared once rates were adjusted for differences in two measures, income and household size.

Although sociocultural factors may differentially associate with health disparities, the latter may not be attributed to socioeconomic status alone. National data show that non-poor African Americans have higher rates of diabetes and obesity than poor African Americans (NCHS 2009-2012). Unhealthy behaviors among different groups (e.g. smoking, excessive alcohol use, poor diet choice) undoubtedly contribute to health inequalities across samples and ethnic groups. In a sample of African, European, and Hispanic Americans living in New York City, African Americans experienced more hypertension and consumed more sodium and less potassium than did European Americans (Bartley et al. 2014). However, even when controlling for health behaviors, health differences between African and European Americans were not eliminated completely (Bartley et al. 2014; Davis et al. 2014; Dressler 1993; Dressler and

Bindon 2000; Hernandez et al. 2014). One suggestion is that individual behaviors along with other factors such as family dynamics may contribute to diversity in adult health.

Sociocultural factors beyond SES and unhealthy behaviors likely contribute to health disparities among USA minority groups. Dressler (1993), for example, argued that ethnic health disparities reflect racism in-color conscious societies. A growing body of evidence suggests African American health disparities may reflect institutionalized racism, which impacts African Americans' access to goods, services, and opportunities (Carlson and Chamberlain 2005; Smedley et al. 2002; Smelser et al. 2001). Such institutional racism is experienced during all aspects of daily life including educational and employment settings, variability in income and SES, and segregation in housing and insurance coverage (Carlson and Chamberlain 2005).

Measuring social stressors like discrimination and their effects on African American health continues to be widely debated, stemming from the inherent difficulty of linking cultural differences to individual health (Carlson and Chamberlain 2005; Dressler and Bindon 2000). Many studies targeting racial discrimination focus on European Americans' perception of African Americans, rather than African Americans experiences of discrimination (Shelton 2000). Conventional biomedical models examine the individual, but emphasize parts, rather than the entire soma (Plsek 2001). A traditional concept is that physiological control is maintained through homeostasis via tightly regulated feedback mechanisms that reduce variability (Carlson and Chamberlain 2005; Seyle 1956; Sterling and Eyer 1988). In recent years, concepts of allostasis and allostatic load based upon neurohormonal interactions were developed to assess the immediate responses to stressors. In contrast to homeostasis, allostasis produces a rapid human physiological stress response based in neurological responses to stressors both physical and perceived (Carlson and Chamberlain 2005; Seeman et al. 1997). Seyle (1956) first hypothesized

that chronic stress may have a cumulative damaging impact on the soma over time. Allostasis is the process by which organisms achieve physiological stability through continual change in response to the current environment (Sterling and Eyer 1988). Allostatic load provides a measure of the overall wear and tear on physiological and somatic systems resulting from adaptive stress reponses to stressors (McEwen and Seeman 1999). Thus, one's allostatic load increases throughout life and a higher allostatic load at any given age indicates poor health and somatic resilience.

Individual perceptions and experiences of adaptive challenges (stressors) impact individual physiological responses to adaptive challenges (McEwen 2002). African Americans experience different stressors than European Americans due to their membership in an ethnic minority. These differences likely influence their risk factor profiles, allostatic load, and health status. Multiple factors along with complex interactions among genes, environment, and culture, rather than any single underlying factor, likely promote observed health disparities between ethnic groups in the USA. Regardless of their underlying causes, health disparities are pervasive and multiple risk factors tend to be elevated in African Americans; we examined components of allostatic load and aspects of body habitus in a sample of middle-class African American residents in central Ohio to explore association of blood pressure, serum glucose, and body habitus with age and sex.

Participants and Methods

Participants

Data were obtained during a study of diabetes and aging in Afircan Americans residing in central Ohio (see Robinson 2003). The original focus for this project was on type II diabetes mellitus and patterns of age-related change. Our participants represent middle-class African Americans residing in Central Ohio. While African Americans are a minority group in Columbus and Xenia, they represent the majority population in Wilberforce Township and a sizeable portion of the Dayton population. In general, members of this sample reported little or no experience of discrimination in their daily lives and almost 70% had at least some college education, with 36% of those holding a bachelor degree or higher (Robinson 2003).

Goals

Based upon existing research, we hypothesize women will show higher blood pressure, glycemia, weight, body mass, waist circumference, trunk-limb ratio, and greater subscapular and suprailiac skinfolds. Conversely, we hypothesize men will show greater height, hip and upper arm circumference, abdominal depth, triceps and medial calf skinfolds, and waist-hip ratio. We hypothesize that body habitus and sex will be significantly and independently associated with blood pressure and glycemia after controlling for age variation. Finally, we hypothesize that this sample of middle-class African Americans will show lower blood pressure, glycemia, and body habitus than previously studied samples of African Americans from lower income and rural populations.

Methods

Multiple anthropometric and physiological variables were assessed on all participants. Age was not self-reported and verified for ten individuals and sex was not recroded for one

individual. Individuals who ate, drank, or smoked within twelve hours of attending the study protocols were not included. Pulse rate, systolic (SBP) and diastolic (DBP) blood pressure were measured three times on the left arm following protocols of the Systolic Hypertension in Elderly Program (Hughes and Schnaper 1982). Participants were given five minutes to relax before and after the first and second pulse and BP measurements (see Robinson 2003 for details of measurement protocols). For analyses, the second and third measured pulse rates and BP measurements were averaged. Systolic and diastolic blood pressures were considered hypertensive at or above 140 mmHg and 90 mmHg, respectively (Chobanian et al. 2003; James et al. 2014).

Following pulse and blood pressure measurements, blood samples were obtained to assess fasting glucose levels. Participants then consumed a 75 g oral glucose load (Orangedex ®, or Koladex ®) and proceeded to anthropometric technicians for somatic measurements. A two-hour post-load blood draw was taken at the conclusion of participation to determine post-load glucose levels. Seven participants with fasting serum glucose levels above 185 mg/dl were advised to seek medical care and did not receive an oral glucose load for measuring post-load glucose. Individuals with fasting glucose levels at or above 100 mg/dl and post-load glucose levels at or above 140 mg/dl were considered at risk for diabetes or diabetic (ADA 2015).

Anthropometric measurements were recorded by two anthropometric technicians following protocols outlined by Lohman et al. (1988). Measured were height, weight, waist circumference, hip circumference, upper arm circumference, abdominal depth, and the triceps, subscapular, suprailiac, and medial calf skinfolds. Weight (lb) and stature (cm) were both measured twice and averaged. All other anthropometric variables were measured to the nearest

millimeter three times and averaged. Subscapular skinfold measurement was not recorded for one individual, but was replaced with the sex-specific average for statistical analyses.

Three indices of body habitus were determined. Waist and hip circumferences were used to calculate waist-hip ratio (w/h):

$$\text{w/h} = \frac{\text{waist circumference (mm)}}{\text{hip circumference (mm)}}$$

Body mass index (BMI), a measure of relative weight, was determined using height and weight:

$$\text{BMI} = \frac{\text{weight (kg)}}{\text{height (m)}^2}$$

Subscapular, suprailliac, triceps and medial calf skinfolds were used to determine a trunk-limb ratio to assess fat patterning:

$$\text{t/l} = \frac{\text{subscapular skinfold (mm)} + \text{suprailiac skinfold (mm)}}{\text{triceps skinfold (mm)} + \text{medial calf skinfold (mm)}}$$

A higher w/h or t/l ratio indicates greater body fat compared to the limbs, while a higher BMI indicates high body weight for height. For analysis, individuals with BMI measurements between 25.0 and 29.9 kg/m^2 were considered overweight and individuals with BMI measurements above 30.0 kg/m^2 were considered obese (USDA and HHS 2010). All measurements and assessments were fully approved by the Institutional Review Board of The Ohio State University.

Statistical Analyses

As a first step in analyses, we determined means, standard deviations (sd) and ranges for all available measures for the total sample and separately by sex (Table 1). For observed differences between men and women, statistical significance was determined using two-sample t-tests with unequal variances. Following the standard medical criteria described in methods, we determined frequencies of overweight, obesity, prehypertension, hypertension, pre-diabteic, and

diabetic individuals in the sample. Next, we determined differences between those with and without diabetes or hypertension using two-sample t-tests with unequal variances for all study variables.

Next, we used hierarchical linear regression to assess independent associations of first sex, then age and sex, and finally age, sex, and each index of body habitus on SBP, DBP, and both fasting two-hour post-load glycemia. Because of the multiple measures of body habitus and their covariation, we also used principal components analysis (PCA) to determine possible composites of age, sex, and body habitus measurements that might better explain variability in blood pressure and glycemia. Last, we compare results observed in this cohort to data reported from other samples of African Americans to determine if this middle-class sample show lower blood pressure, glycemia, and body habitus than observed elsewhere.

Results

Contrary to our hypothesis, men were significantly heavier and showed higher DBP and a greater t/l than did women (Table 1). Additionally, women showed significantly larger triceps and medial calf skinfolds than men. As hypothesized, men were significantly taller and showed a higher w/h than women. Men and women showed no significant differences in means (sd) or ranges for systolic blood pressure, pulse rate, fasting or post-load glucose, BMI, abdominal depth, subscapular or suprailiac skinfolds and waist, hip, or upper arm circumferences. Although not statistically significant, men showed higher SBP and upper arm circumferences, while women showed higher pulse rates, BMI, and fasting and post-load glucose levels and showed greater hip circumference and subscapular skinfolds (Table 1).

Following standard criteria listed in methods, 73% of this sample are pre-diabetic or diabetic based on fasting glucose, yet even when including participants with fasting glucose levels too high to receive an oral glucose load, only 36% of this sample are considered at risk for or with diabetes based on post-load glucose (Table 2). About two-thirds of this sample are prehypertensive or hypertensive based on SBP, yet only half are prehypertensive or hypertensive based on DBP (Table 2). While 32% of participants have systolic hypertension, only 12% are considered hypertensive based on both SBP and DBP (BP $\geq$ 140/90 mmHg). Almost 80% of this sample are considered overweight or obese (Table 2).

Based on fasting glucose levels, 71% of men are at risk for or have diabetes compared to 74% of women. However, based on post-load glucose levels, this falls to 32% and 37% for men and women respectively (Table 2). Blood pressure showed the largest discrepancy between men and women in this sample. While 80% of men are prehypertensive or hypertensive based on SBP, only 60% of women met thiscriteria (Table 2). Additionally, 86% of men are overweight or obese, whereas 76% of women. Sampled women have a greater burden of diabetes, systolic hypertension, and obesity than did men (Table 2).

In bivariate analyses, age, SBP, DBP, pulse, height, abdominal depth, BMI, w/h, and both subscapular and suprailiac skinfolds are associated significantly with elevated post-load glucose (Table 3). Only SBP, subscapular skinfolds, and abdominal depth are associated significantly with elevated fasting glucose (Table 4). Age, post-load glucose, abdominal depth, and both waist and hip circumferences are significantly associated with systolic hypertension (Table 5). No measure significantly associated with diastolic hypertension in the total sample (Table 6).

Bivariate Pearson correlations between all measures in this study are presented in Table 7. When explained using bivariate linear regression, age and BMI are account for a significant

amount of variation in SBP and post-load glucose, but not DBP or fasting glucose (Table 8). Similarly, w/h significantly predicts SBP, fasting, and post-load glucose, but not DBP (Table 8). Of all the variables examined, only t/l was not associated significantly with any dependent measure (Table 8).

In multivariate analysis, the joint effects of age and sex on outcomes both are independently predictive of SBP and post-load glucose (Table 10). When controlling for age and sex, BMI maintains a significant and independent predictor of SBP and post-load glucose, while w/h is a significant predictor of not only SBP, but also both fasting and post-load glucose levels (Tables 9 and 10). When adjusted for age, the t/l is significantly and independently associated with SBP (Table 9). As a further step in this analysis, all biomarkers associated significantly with outcomes in bivariate analyses at $p \leq 0.15$ were entered into multivariate regressions as independent variables with glycemia and blood pressures as dependent variables. In these analyses, age was a significant independent predictor of SBP ($p=0.03$). Height was a significant independent predictor of both DBP ($p = 0.028$) and post-load glucose ($p=0.023$), but only w/h significantly and independently predicted fasting glucose ($p=0.047$), although the latter showed an almost significant association with post-load glucose ($p=0.063$).

To further explore possible relationships between body habitus with outcomes, we utilized principal components analysis (PCA) to determine body habitus composites that might better account for variability in blood pressure and glycemia. All anthropometric measurements and indices, but not age, were entered into a PC model without rotation. Components with eigenvalues >1.0 were extracted. Jointly, the first three component factors account for over 75% of total variance in these body habitus biomarkers (Table 11). Based on logical consistency, we described the first component (PC 1) as “overall body habitus,” the second component (PC 2) as

"trunk/limb fat patterning", and the third component (PC 3) as "waist/hip fat patterning." Factor scores were generated to measure each participant's position on the spectrum described by each PC. These factors scores were used in bivariate and multivariate regression analyses as independent variables with blood pressure and glycemia as the dependent variables.

PC 1 (overall body habitus) significantly and independently predicts both SBP (p=0.016) and post-load glucose (p = 0.001), even when controlling for age and sex (Table 12 and 14). Similarly, PC 2 (trunk/limb fat patterning) significantly and independently predicts both SBP (p = 0.011) and DBP (p=0.017) when controlling for age alone, but not when controlling for both age and sex (Table 13). PC 3 (waist/hip fat patterning) is a significant predictor of both fasting (p=0.038) and post-load (p=0.005) serum glucose levels (Table 14). Controlled for age, PC 3 shows a significant and independent association with both DBP (p=0.045) and post-load glucose (p=0.002) and a borderline association with fasting glucose (p=0.060) (Table 13). With control for both age and sex, PC 3 only significantly predicts post-load glucose (p=0.002) and retains its borderline association with fasting glucose (p=0.059) (Table 14).

Discussion

Overall, this sample of middle-class African Americans is overweight and at high risk for diabetes and hypertension. Based on fasting glucose levels, over half the sample is at risk for diabetes and about one fifth have diabetes. However, when using post-load glucose levels, those percentages drop to 20% and 11% respectively. Results support our hypothesis that women show poorer aspects of body habitus and higher serum glucoses levels than men. Women in our sample show more fat distribution on their limbs and trunks, and exhibit higher BMI, hip circumference,

and both fasting and post-load glucose than men. Contrary to our hypothesis, men show higher blood pressure (SBP and DBP) than women in this sample.

It is clear that aspects of body habitus significantly associate with chronic conditions in our sample. Specifically, fat located in the thoracic and abdominal trunk regions associate with elevated post-load glucose, fasting glucose, and SBP. Both BMI and w/h significantly and independently predict SBP and post-load glucose in our sample. Similar to European Americans, BMI and other measures of body habitus are associated with elevated blood pressure and glycemia in our sample (Zhang et al. 2009). Despite these similar associations between aspects of body habitus and physiological outcomes, our sample shows higher rates of obesity, hypertension, and diabetes than do European Americans on a national scale (Table 15). While African and European Americans share similar biological risk factors for chronic conditions, it seems likely sociocultural factors are responsible for higher incidences of chronic conditions among African Americans in our sample and nationally. In addition, our middle-class African American sample shows about the same frequencies of pre- and full hypertension (66.7%) as the national average (65%) as well as overweight and obese (sample: 78.8%, USA: 75.9%).

Table 15. Rates of diabetes, prehypertension, hypertension, overweight, and obesity among African and European Americans in the United States.

Chronic Condition	African American	European American	Sample Totals
Diabetes	18.3	9.2	19.8/11.0 (Fasting/Post-Load)
Prehypertension	22.2	13.2	35.1
Hypertension	42.8	24.7	31.6
Overweight	27.6	33.5	40.7
Obesity	48.3	33.3	38.1

(Source: NCHS 2009 – 2012)

Contrary to our hypothesis, when comparing our sample to less well off African Americans nationally, they show higher incidences of diabetes, prehypertension, and overweight than expected (Table 16). However, this middle-class sample does show lower rates of full-blown hypertension and obesity than poorer African Americans. These trends seem to indicate that our sample contains a larger proportion with blood pressures and body sizes in the prehypertension and overweight classes than observed in the NCHS 2009-2012 data. This may reflect less risk for clinical hypertension and obesity in our sampled compared to poorer African Americans. Yet, a closer look beyond national survey data reveals that our sample is more overweight and obese than rural poor African Americans, while rural African Americans show much higher rates of hypertension (Table 17).

Table 16. Rates of diabetes, prehypertension, hypertension, overweight, and obesity among Poor African Americans in the United States.

Chronic Condition	Poor African Americans	Sample Totals
Diabetes	17.9	19.8/11.0 (Fasting/Post-Load)
Prehypertension	25.3	35.1
Hypertension	44.7	31.6
Overweight	25.4	40.7
Obesity	48.7	38.1

(Source: NCHS 2009 – 2012)

Table 17. Rates of diabetes, hypertension, overweight/obesity among rural poor African American men and women in Georgia and our sample.

	Rural African American Men	Sample Men	Rural African American Women	Sample Women
Diabetes	11.7	17.1/8.6 (Fasting/Post-Load)	15.8	21.8/10.3 (Fasting/Post-Load)
Hypertension	51.5	28.6	50.8	33.3
Overweight/Obese	79.4	85.7	74.2	75.6

(Source: Davis et al. 2014; Quarells et al. 2012).

While our sample shows higher rates of chronic conditions and risk factors than European Americans on a national level, the differences among African American samples themselves are more difficult to interpret. That fasting glucose, two-hour post-load glucose, BMI, and waits/hip ratio are near or above cut-points suggesting diabetes and obesity in the middle-class African American participants in our study indicate that these conditions affect

them as they do lower-class African Americans. However, this African American sample with higher socioeconomic status shows less clinically diagnosed hypertension and diabetes. We suggest that these differences may reflect sociocultural differences between settings and a need for greater attention to risk factors among poorer and rural African Americans.

Limitations

Results from this study provide corroborating evidence that aspects of body habitus and physiology may differ across groups of African Americans by social class. However, several limitations affect this study. First, although study sample was obtained in central Ohio, it is not likely to be representative of even this area, let alone all middle-class African Americans in the USA. Secondly, these data were initially collected as part of a study on type II diabetes mellitus. This may have encouraged participation by individuals previously diagnosed with diabetes or who wished to assess their current health condition. Furthermore, although the sample is sufficient for analyses presented here, these are potential biases. Additionally, blood pressure and serum glucose levels offer only a single snapshot of an individual's physiological function at a moment in time. This is most evident in the disparities between those considered pre-diabetic or diabetic based on fasting glucose levels (73.2%) and post-load glucose (31.2%) in our sample. Blood pressure and glycemia vary among individuals and between populations. For example, an individual with an average fasting glucose level at or above 126 mg/dL may actually show glucose levels ranging from 112 – 140 mg/dL over a day (Sacks 2011). Recognizing that serum glucose levels can be highly variable, the American Diabetes Association (2015) recommends doctors repeat serum glucose measurements 3-6 months after initial diagnosis of pre-diabetes/diabetes. In our study, no follow up tests were conducted to determine the consistency

these measures over time. Lastly, we are limited by the specific physiological data available and a lack of data on sociocultural factors. As previously mentioned, this study includes several components of allostatic load, yet it lacks several others. Thus, we were unable to fully assess physiological wear and tear within our sample or to document sociocultural experiences of these middle-class members of the African American community in the USA.

Conclusion

Despite possible limitations, our sample shares similar risks for chronic diseases as do European Americans, but they also experience a heavier burden of hypertension, obesity, and hyperglycemia indicating the likelihood that sociocultural factors contribute to observed ethnic health disparities. Relationships among health, socioeconomic status, and ethnicity are not yet fully understood. Incorporating more social variables into future analyses likely will provide deeper insight on these health disparities and allow us to test hypotheses based on cultural practices and behavior (e.g. discrimination, diet choices).

Undoubtedly, multiple factors and complex interactions among genes, environment, and culture rather than any one underlying factor are causally related to observed health disparities. Considering the appropriate theoretical approach to understanding these multiple factors and their complex interactions is a difficult task. By experiencing specific stressors related to membership in an ethnic minority, African American health status may in part be determined by specifically socioculturally generated stressors. Only by determining the different stressors impacting African American daily life will we be closer to understanding why and how ethnic health disparities develop and persist.

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